

ON CERTAIN RELATIONSHIPS
BETWEEN PLEURISY AND
TUBERCULOSIS.

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C O N T E N T S.

	Page.
<u>Part I.</u> On the True Nature of Pleurisy	1-47.
<u>Part II.</u> Certain Observations on cases of Pulmonary Tuber- culosis which started from a pleuritic origin.	48-61.
<u>Conclusion.</u>	61-64.
<u>Bibliographical Reference</u>	65.

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PART I.

ON THE TRUE NATURE OF PLEURISY.

There is perhaps no part of medical practice which gives the practitioner more trouble or anxiety than the stating of a prognosis. The wise man, as a rule, is content to be so vague that his information is of little value; it is only the rash who is clearly definite. And if this be so as regards an immediate prognosis, the difficulties which surround the enunciation of a remote one are ten times greater, though in a sense, the latter is more important than the former. For if a man be acutely, dangerously ill, we are content to wait to see what each day shall bring forth, indeed we have no option; but if the disease be chronic, above all if it be, at the time in question, seemingly slight, what a number of difficult questions arise: shall the patient work? if so, what kind of work? when may he begin? is his life insurable? and so on.

These questions are difficult to answer and the

remote prognosis is difficult to state, because we are ignorant. It is in the hope that some of this ignorance may be cleared away in the case of one disease that this thesis is put forward.

The idea that pleurisy is to be regarded as predisposing to pulmonary tuberculosis is not new. In 1815 Thomas Young said: "Peripneumony, when bleeding has been too sparingly employed, frequently lays the foundation of consumption." This teaching, however, was lost sight of for a time, and so late as 1856 Stokes does not doubt that primary pleurisy a frigore is really primary and simple.⁽²⁾ Of later years a reaction has set in, and the majority of writers at the present time regards most pleurisies as manifestations of tubercle. Thus: "A⁽³⁾ considerable proportion of cases of pleurisy, dry and serous, are tuberculous. This fact must at once arrest the attention of the practitioner. His patient is the subject of a disease of the chest that will invade the lungs, either now or at any time, or at different times in the future. Pleurisy, haemoptysis, local tuberculosis in regular or irregular succession con-

(1) A treatise on comsumptive diseases, by Thomas Young. 1815.

(2) Stokes on Diseases of the Chest. Dr Hudson. New Sydenham Society. 1882. Page 490 et seq.

(3) The Principles of Treatment. J. Mitchell Bruce. First Edition, p.400.

stitute the main features of the natural history of pulmonary tuberculosis in many instances." This may be taken as a fair example of the most usual modern view of the situation. "There are, however, writers who believe that all pleurisies are tuberculous, and there is still at least one of note, who does not think that there is much connection between the two conditions. The belief of the latter is that "there⁽¹⁾ is no reason to think that simple pleurisy disposes to phthisis." Those on the other hand who believe that all pleurisies are tuberculous are equally emphatic. "J'ai, l'appuyant sur une série de preuves, énoncé et défendu cette opinion, que la pleurésie dite a frigore, que la pleurésie à grand épanchement, à allures febriles, à début soudain, éclatant après refroidissement chez un sujet regardé comme vigoureux et bien portant, devait être suspectée manifestation de tuberculose Tout ^{individu} ~~individu~~ qui ne peut fournir pour raison de son épanchement, ni une infection (scarlatine, puerperisme etc., etc.), ni une dyscrasie (rhumatisme), ni un trauma (fracture de côte, infarctus pulmonaire), cet individu est un tuberculeux, fut-il vigoureux, jeune, gros, et gras, se déclarât-il bien portant et indemne d'antécédents phymateux hereditaires ou personnels." (2)

(1) Percy Kidd: Syst. of Medicine, Edited by Clifford Albutt, Vol.V. p.164.

(2) Revue de Medicine: Tome VI. Juillet, 1886. pages 611 and 612. "De la pleurésie dite a frigore" L.Landouzy.

In view then of these differences, I propose to put forward certain facts, which have come under my own observation, and further to state the results obtained from certain statistics to which I have had access, but which have not hitherto been published. I intend also to refer to certain published figures.

To determine whether pleurisy is to be regarded as a real fore-runner of pulmonary tuberculosis, two methods of clinical investigation are open. We may trace out the ultimate histories of a large number of cases of pleurisy; or we may seek for a former history of pleurisy in a large number of patients who come presenting conditions of pulmonary tuberculosis. The first of these methods is obviously the better, but the more difficult to accomplish seeing that it cannot be regarded as complete till every pleuritic in the list has died, a process which may occupy many years. I propose, however, to take up the study from this aspect first.

There are not many observers who have worked out this matter, but those who have, have come to very definite conclusions, viz., that there is very strong evidence in support of the view that many pleurisies are tuberculous, and also that many are not.

In this connection we ought to clearly define what variety of pleurisy it is of which we are speaking. As regards dry pleurisy I have not a great deal to say. Dry pleurisy is without doubt a very frequent disease often overlooked, so often that it is impossible to say what is the usual remote event after its occurrence: certainly it cannot be clinically recognisable tuberculosis, else tuberculosis would be commoner than it is; post mortem examination also reveals adhesions much more frequently than could be the case if all these pointed to tuberculosis. "The ⁽¹⁾ thoracic cavities have been repeatedly found all closed by old adhesions when there had been no history of any chest affection." Again, "Adhesions ⁽²⁾ of the entire pleural surface have been found post mortem in the bodies of persons who never have been seriously ill."

Sir Andrew Clark regarded dry pleurisy as the sole cause of origin of what he called "fibroid phthisis", an affection which he believed to be non tuberculous. ⁽³⁾ This condition was admitted by him to be one of great

(1) The Principles and Practice of Medicine by Hilton Fagge. Second Edition, 1888. Vol.II. Footnote to page 179.

(2) A Manual of Medical Treatment. I. Butney Yeo. Fifth Edition. Vol.I. page 601.

(3) Fibroid diseases of the Lung. 1894. Page 82.

rarity, and indeed there seems to be some doubt as to what its pathology actually was.

It is more particularly with pleurisy with effusion that we have to deal. Here much more definite data are available. The disease is one which presents such striking features that it must be rare, that it is overlooked by the patient. Latent effusions do, of course, exist, but usually at some period of the illness the attention of the patient is drawn to his condition of health, and advice is sought. Here let me recapitulate. The tendency at present is to regard the majority of cases of pleural effusion as tuberculous; the former belief was that they were due to chill - an aetiological factor which has of late received a perhaps unmerited amount of scepticism. A young man walked home one cold winter night in light clothes after being heated by a vigorous dance. Next day he was seized with pain in the left side, and in a few days the whole left chest was found to be nearly full of fluid, which in the course of a few weeks subsided. This person remained in perfect health for three years when he died of diphtheria. It is difficult to see why such a case should of necessity be regarded as tuberculous, or even organismal. It is still believed that the temporary febrile state induced in some nervous men by the passage of a bougie is not organ-

ismal - it can be prevented by the use of a local anaesthetic - and if this be true we have then as established the fact that fever may be non microbic.

It has been shown that in the horse, pleurisy a frigore exists, and that in flocks of sheep after clipping in cold weather it is common enough; several of these cases were examined post mortem and no evidence of tuberculosis was found.⁽¹⁾

However, as has been said the tendency is to regard most cases of pleurisy with effusion as tuberculous. In this connection valuable statistics have been published by Bowditch, by Barrs, by Osler, by Kingston Fowler, and very recently by Salanoue-Ipin and by Hedges.

Barrs⁽²⁾ collected between the years 1880 and 1884 one hundred and fourteen cases of pleural effusion of which 74 were sero-fibrinous and 40 were purulent. Of the 74, 32 were dead by the year 1890, 25 were alive, and 17 had disappeared from observation. Of the 32 dead, at least 17 had died of tuberculosis, possibly more. This disease therefore accounted for more than one half of the deaths; and it attacked nearly one fourth of all the patients who had had pleurisy with effusion - at least one fourth of the cases of sero-fibrinous effusion ultimately became

(1) Archives de Medecine Navale. April 1900. P.261

(2) Alfred G. Barrs, M.D. British Medical Journal. May 10th, 1890.

tuberculous. Of the 40 cases of purulent effusion it was not proved that any became tuberculous.

Salanoue-Ipin⁽¹⁾ collected a total of 352 cases of sero-fibrinous effusion from the records of the Naval hospital at Brest, between the years 1877 and 1889. Of these 131 are now dead. In 84 of these death was due to some affection tuberculous in nature. It does not follow as will be shown subsequently that in the remaining cases this high proportion should be maintained: it is much more likely that in the rest a smaller rate of deaths will be due to tubercle, so that it would not be right in the least to say that a percentage might be found by stating that 84 pleurisies out of 131 are tuberculous.

Osler⁽²⁾ analyzed the post mortem records of 101 cases of pleurisy, fibrinous, sero-fibrinous, haemorrhagic and purulent, and found that of these 32 were definitely tuberculous, i.e. in them there were found miliary tubercles, caseous masses or fibro-tuberculous membranes. These 101 cases were not, however, all cases of pleurisy a frigore: many were terminal pleurisies which hastened the fatal event in chronic cases of kidney, cardiac, hepatic

(1) Pronostic éloigné des pleuresies séro-fibrineuses. Archives de Médecine Navale. Avril 1900. P.265.

(2) The Shattuck Lecture on Tuberculous Pleurisy. Mass. Medical Society, 1893. Pages 3 & 4.

or other disease; in some of these even the pleurisy was distinctly tuberculous. This important fact, viz. that the terminal pleurisies in chronic non-tuberculous visceral disease are themselves frequently tuberculous in nature has been further proved and insisted on by Kingston Fowler⁽¹⁾.

Bowditch,⁽²⁾ Junior, published the results of 90 cases of pleurisy of which 30 died of pulmonary tuberculosis. He did not, however, distinguish between the forms of pleurisy, so that the value of his paper is not so great as it would have been if this had been done.

Kingston Fowler⁽³⁾ quotes from a number of sources on this point. Out of 326 cases of pleurisy where the subsequent histories were obtained, 181 became tuberculous. This is a percentage of 55.4.

Wilson Fox⁽⁴⁾ mentions 28 fatal cases of pleurisy of which 9 were tuberculous. He believed that in most of these, pulmonary tuberculosis had existed first, and that there had been a subsequent infection of the pleura.⁽⁵⁾

(1) Diseases of the Lungs. Fowler & Godlee. 1898. Page 544.

(2) Medical News. 1889. Vol.LV. Page 63.

(3) Diseases of the Lungs. Fowler & Godlee. Page 545 et seq.

(4) Treatise on Diseases of the Lungs & Pleura. Wilson Fox. Page 1094.

(5) Ibid, Page 538.

Hedges⁽¹⁾ collected 130 cases and stated that 56 of these became tuberculous. Nine of these, however are very doubtful and certainly ought not to have been included in his list of tuberculous cases. This gives us 47 which certainly became tuberculous - a percentage of about 36. One of his doubtful cases is a fair example of the remainder⁽²⁾. A man was tapped three times for pleurisy with effusion. Six months later he developed a cough, had occasional night sweats, and occasional creaks at the left apex. But it should be observed he had had influenza three weeks before, a disease which is without doubt often accompanied by or followed by signs of catarrh at an apex, a catarrh which does not seem very liable to become tuberculous.

Of all these statistics the most pertinent to our present enquiry are those of Salan^ude-Ipin, of Barrs and of Hedges. They alone have clearly differentiated the sero-fibrinous cases from the others. It is certainly a serious error to mix up dry, purulent, haemorrhagic and sero-fibrinous pleurisy and draw a statistical deduction therefrom.

I have said that the pleuritics who become tuberculous do so early, within a few years: and that if they escape tuberculosis for a few years they

(1) St. Bartholomew's Hospital Reports for 1900.
Vol. XXXVI. Page 83.

(2) Ibid. Page 96. Case 4.

are not very likely to develop that disease later. Thus Barrs had 74 cases of sero-fibrinous effusion, of which 17 died of known tubercle: 15 others died of non-tuberculous conditions. That is to say that 23 per cent of the cases of pleurisy became tuberculous, and that rather more than 50 per cent of the deaths after a number of years were due to tuberculosis. ^MMy belief is that when the remaining 42 have died, it will not be found that 50 per cent of them have died of tubercle, but a very much smaller number; and that out of these pleuritics 23 per cent is a better estimate of the case mortality of the whole due to tubercle, than is 50 per cent. The proof of this suggested itself while I was studying some statistics which were kindly put at my disposal by three Scottish insurance offices, whose names I regret I am not at liberty to disclose. My line of enquiry was to go over a large number of proposal papers, mark those which had given a previous history of pleurisy, and then if the policy had terminated by death, to refer to the death certificate for the cause of death. This investigation was not calculated to throw much light on the percentage of incidence of tuberculosis after pleurisy, for it is obvious that all cases, which had developed tuberculosis shortly after a pleurisy, would be rejected by the examining physician, and so would not occur on

the books of the company. Other difficulties soon presented themselves during my work. A large number of policies are allowed by their holders to lapse, so that in this way much valuable medical information is lost. Again the history of pleurisy is one so seldom occurring among candidates for insurance - accepted candidates at least - that an enormous number of proposal forms had to be gone through, nearly twelve thousand in all. Out of these only thirty cases of pleurisy were obtained.

Nevertheless some useful lessons may be learned from the study of these. I propose to divide them into two lots, as one office had obviously not been so careful to reject pleuritics as had the other two. In this first office 685 forms were examined, and I was able to find 14 cases of pleurisy; while out of the eleven thousand forms examined in the other offices only 16 cases of pleurisy were found altogether.

Let me deal with the latter set of cases first. About eleven thousand policies were examined; in 16 there was found a history of previous pleurisy. Of these 16, three only died of pulmonary tuberculosis; in 13 the cause of death was other than tuberculous. Of the three tuberculous cases, the pleurisy in one occurred only one year before the proposal, and the death four years later; in the second the pleurisy

occurred eight years before the proposal, and the death four years later; in the third the pleurisy was followed in four years by an haemoptysis, in ten years by the acceptance of the life with a query as to the pulmonary state, and one year later by death. In the other thirteen, non-tuberculous cases the proposal was in most cases at an interval longer than ten years from the date of the pleurisy; and in all but two, the date of death was very much more than ten years after the pleurisy had occurred. The following is a tabular statement of the cases.

Age at Pleurisy.	Age at Proposal.	Age at Death.	Cause of Death.
33	34	38	Pulmonary tuberculosis.
16	24	28	"
14	Haemoptysis at 18. 28	29	"
10	19	30	Not Tuberculosis.
35	38	56	"
37	40	42	"
14	29	52	"
19	32	33	"
?	50	61	"
18	25	31	"
19	31	36	"
20	31	44	"
28	30	42	"
22	42	59	"
23	27	46	"
43	59	71	"

In the other set of 14 cases, 8 became tuberculous. In all these but one, the interval of time between the pleurisy and the proposal was under four and a half years, while in the case of those who did not become tuberculous, four had their policies effected at an interval of more than ten years after the attack of pleurisy, and only two after a shorter interval. A table of these cases similar to the above is subjoined.

Age at Pleurisy.	Age at Proposal.	Age at Death.	Cause of Death.
24	28	31	Pulmonary Tuberculosis
32	34	36	"
26	30	36	"
25	28	39	"
33	34	40	"
26	30	48	"
31	34	38	"
23	38	42	"
49	49	50	Not Tuberculosis,
30	35	36	"
12	32	35	"
23	41	80	"
19	39	45	"
18	45	49	"

These two sets of figures suggest that there is a greater expectation of an individual becoming phthisical if he has had a recent pleurisy, than if he had had a pleurisy many years ago. The practical lesson from an insurance point of view is, that no life ought to be considered, which has a history of an attack of pleurisy of less than five years standing; that after this time has elapsed the life should be rated up; and that this rating up should vary inversely as the number of years since the pleurisy has occurred.

Shortly after studying these cases my attention was drawn to the valuable paper of Salanoue-Ipin, already mentioned. In it I found a further proof as regards this matter. In his cases in addition to the 84 who, he knew, died of tuberculosis, he claims as tuberculous 19 who died, and where the cause of death was unknown. In some obscure way he supposes that these latter would be found to have died of tuberculosis had he only been able to get at the certificate! He does this without much proof of his right to do ^{so} ~~as~~. Still the result he gives is most interesting. Out of the 103 thus obtained, 33 died during the first year, 10 during the second, 6 during the third, 8 in the fourth, and so on till the tenth year, by which time 92 had died. The remaining eleven died during the second decade.

"Enfin, au bout de vingt ans, le bacille de Koch a terminé son oeuvre"(1), and the cases remaining were non-tuberculous.

My special reason for bringing forward these sets of statistics now, was to show that observers who have watched a number of pleuritics for fifteen or twenty years, and noted that a certain number of them have died of tuberculosis, may almost certainly state this number as the total number of those pleuritics, which ever would become tuberculous. Both my insurance figures and these last of Salanoue-Ipin tend to prove this. Therefore we may fairly take the figures of Barrs, of Salanoue-Ipin and of Hedges as they stand; and from them we may arrive at a figure which will show what percentage of cases of sero-fibrinous pleurisy ultimately develop tuberculosis. As has been already stated, in Barrs' cases 17 out of 74 became tuberculous, in Salanoue-Ipin's 84 out of 352. Both these equal 23 per cent. In Hedges cases 47 out of 130 became tuberculous; this is 36 per cent. The conclusion which I wish to state is, that if a large number of cases of primary pleurisy with effusion be taken, it will be found that from one fourth to one third of these will ultimately develop tuberculosis; and that in the vast majority, if this event is to take place at all, it will have done so within ten years from the date of

(1) Salanoue-Ipin. Ibid, page 266.

the pleurisy. After this time the patient is comparatively safe.

It is certainly unfortunate that there are no statistics which embrace the ultimate event in a whole set of pleuritics; but the difficulties in the way of accomplishing this are manifestly enormous; indeed it could only be done by someone who inherits not only the case-books, but also the patients of a previous observer. Bowditch came nearer to this than any other, but unfortunately he is far from clear as to his differentiation between the various forms of pleurisy.

As regards pleurisy where there is an effusion whose nature is other than sero-fibrinous I have little to say.

Haemorrhagic effusion is usually regarded as of grave omen, pointing either to tubercle or to malignant disease; but of it I have no personal experience.

Purulent pleurisy is certainly much more frequently due to organisms other than the tubercle bacillus. In children under seven years of age this is almost always the case⁽¹⁾. In adults, while the

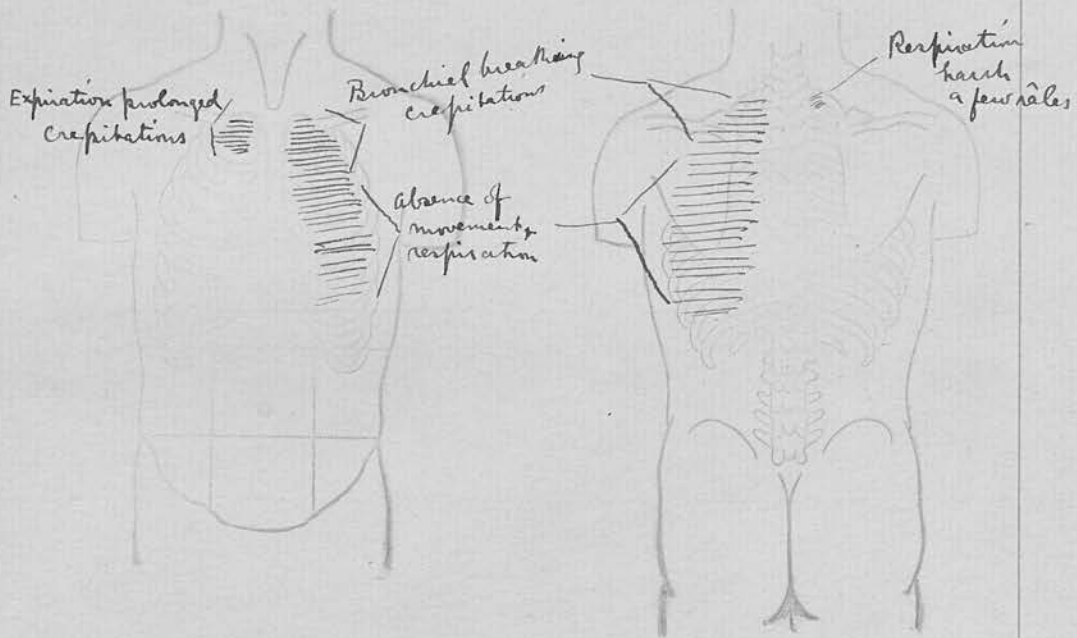
(1) Diseases of Infancy and Childhood. Holt, 1899. Page 552.

condition of purulent tuberculous pleurisy is not unknown, it is most commonly the result of pneumothorax⁽¹⁾. I have seen one case of empyema - operated on before it came under my notice, but still with a sinus - where tubercle bacilli were found in the pus.

This patient, a girl, aged 17 years, was seen by me in October 1900. Her story was, that she had enjoyed good health till May 1899, when, after a wetting she was seized with pain in the left side, for which she went into the Brompton Hospital. She was kept in several months, and in October 1899 was tapped. Thirty ounces of sero-fibrinous fluid were drawn off. She remained in poor health till June 1900, when she again felt her left chest become painful and even noticed that it bulged. For this an operation was performed in June. The sinus was still discharging when I saw her in October; in this discharge, which consisted of a very thick tenacious pus, I found tubercle bacilli without difficulty. Ever since November 1899, i.e. shortly after the first tapping she began to cough and expectorate. These symptoms persisted, and in the sputum also, tubercle bacilli were in abundance when I saw her. At this time also there was great re-

(1) The Shattuck Lecture. Osler, 1893. Page 5.

traction of the left chest, with signs of pulmonary mischief at both apices, especially the left as shown in the accompanying charts.



It is, of course, possible that this patient may have had a pneumothorax, but there was no history at any time of sudden pain or collapse, nor when I saw the patient were there any of the special signs of that condition; so that we may, I think, look on this as a case of purulent tuberculous pleurisy, not due to pneumothorax: and it would indeed be hard to say whether the apical tuberculosis of the lungs or the basal tuberculosis of the pleura were the primary focus of infection.

Osler⁽¹⁾ who writes on this subject with greater clearness than any other author, states that in purulent tuberculous pleurisy, apart from pneumothorax, the effusion is of a milky opalescent character and that it is usually sterile; which is certainly very different from what was found in the case just reported.

Now that it has been definitely shown that a number of cases of sero-fibrinous pleurisy are followed by tuberculosis, and that such total at least one quarter of the cases, it becomes very important to know whether we have at present any definite means of differentiating the tuberculous from the non-tuberculous cases. It is obvious that if a pleurisy with effusion is to be regarded as the fore-runner of pulmonary tuberculosis, its treatment must be conducted on very different lines from what would be right and proper if it were only a local affection. In the latter case the affair would be one of a few weeks at the outside; our main objects would be to get up the general health and to see that the lung expanded. But if the tuberculosis view were entertained, the mere expansion of lung would surely be of very secondary importance: and at this point I fear I must diverge from my line of enquiry as to

(1) The Shattuck Lecture. Page 63.

whether we have any certain sign of tuberculosis, in order to discuss fully the subject of tapping pleural effusions.

At the present time most authorities are in favour of what may be called very early tapping. "No⁽¹⁾ considerable effusion should under any circumstances be left for more than three weeks, unless signs of ebb have commenced and are progressive. Fourteen days would probably be a wiser limit."

"Serum⁽²⁾ should be removed by paracentesis in all cases which present an effusion so great as to fill the pleura, or which are attended by any distress of breathing, or which show no signs of being absorbed after a week or ten days." "When⁽³⁾ however, after an illness lasting from ten days to a fortnight a considerable effusion is present and the pyrexia continues. the question of paracentesis arises if there is still no sign after some days that absorption is taking place it is generally better to withdraw as much fluid as will flow through a syphon aspirator."

(1) The Principles of Treatment by J. Mitchell Bruce. 1899. Page 403.

(2) A System of Medicine. Ed. by Clifford Allbutt. Vol. V. Article by S. Gee. Page 373.

(3) The Diseases of the Lungs. Fowler and Godlee, 1898. Page 574.

All these advocates of early tapping were, however, quite outdone by Potain who advised paracentesis on the second or third day.⁽¹⁾

Now both Kingston Fowler⁽²⁾ and Osler⁽³⁾ have shown that in many cases there are actual tubercles either in the visceral pleura itself, or just beneath it; and Osler believes that the tubercle bacilli are in the subserous lymph spaces of the lung. In this case, it is obvious that the best thing that can happen to this lung is that it should be compressed by serous effusion. By this device it is put to rest; and rest is the greatest curative agent against tubercle. But not only is the part put to rest, but the actual cubic extent of the tuberculous patch will be diminished, and consequently it will be easier for it to be enclosed in a fibrous capsule which might shut it off from the organism for good. How absurd it seems that at the end of a fortnight or three weeks we should upset all this arrangement, make the lung work, break down fibrous bands and possibly shower bacilli over the system.

Further it is possible that the effusion itself is antituberculous in nature, a reaction on the part of the organism attacked by a tuberculous process.

(1) A Manual of Medical Treatment by I. Burney Yeo.
Fifth Edition. Revised. Vol.I. Page 610.

(2) Diseases of the Lungs. Pages 553 to 581.

(3) The Shattuck Lecture.

The pleuritis is not the mere result of mechanical irritation due to tubercles on the pleural surface. Other mechanical stimuli, such as cancer nodules, do not cause a pleuritis though they may an effusion passive in character. Cancer is a disease against which the organism does not seem to attempt to defend itself by inflammatory processes; tubercle is a disease against which the organism does direct inflammatory action, often with the greatest success. Indeed, advanced tubercle can be cured only by a process of inflammation with the formation of new fibrous tissue. We should, therefore, always be careful not to spoil any inflammatory process in the presence of tubercle.

I have myself seen ten cases of pulmonary tuberculosis where the patient had been quite a healthy individual till seized with pleurisy with effusion, apparently a frigore. Of these five were tapped, and five were not. Of the first there was immunity from pulmonary mischief in one case, for four years, but in all the others the pulmonary disease seemed to have been clinically continuous with the attack of pleurisy. Of the five who were not tapped there were in three, periods of immunity of 19 months, 12 months and fifteen years. These figures are few, but suggestive. A close study, however, of the

cases published by Hedges⁽¹⁾ was found to throw additional light on this matter. In his cases of pleurisy with effusion he notes whether tapping was done or not in 128; 64 were tapped and 64 were not. Of the first, 32 developed tubercle and 32 did not. Of the 64 where no paracentesis was performed only 15 devoped tuberculosis and 49 remained free. It is at least indubitable that the greatest incidence of tubercle was among the tapped cases. I have reckoned among the tapped, all those where any fluid whatever was drawn off; in four this was only a few drams, but in all the others it amounted to several ounces.

If we do not tap we run certain risks, viz. a contracted side with scoliosis, an adherent lung apt to ache, permanent shortness of breath, or even sudden death. The first three are of little importance when compared to an attack of pulmonary tuberculosis: it is not acknowledged by all the best authorities that they are likely events.⁽²⁾ The danger of sudden death is a real though happily a rare possibility in cases of very large effusion. I have myself seen two. One was a man of 52, who

(1) St.Bartholomew's Hospital Reports for 1900.
Pages 95 to 145.

(2) "The probability of fibroid changes in the lung as a frequent sequel even in pleural effusions of one or two months standing is very small."
Wilson Fox. Diseases of the Lungs & Pleura.
Page 1052.

having been previously tapped declined further interference. He died while turning over in bed. His right chest was quite full of fluid with considerable cardiac displacement. Post mortem a very firm white ante-mortem thrombus was found extending from the right auricle through the ventricle into the pulmonary artery.

The other was a man of 58, who was tapped on fourteen occasions, nine times by myself. He also died quite suddenly while in bed. The post mortem conditions were similar to the last. Such cases are not, however, common. Wilson Fox says that the "dangers of sudden death in acute uncomplicated pleurisy are comparatively small."⁽¹⁾ One would not, of course, leave these cases of large effusion alone; but it is not necessary to do more than obviate the tendency to death; only a little fluid need be drawn off, and the practice of attempting to empty the pleura cannot be too strongly condemned. Luckily nature is frequently willing and able to mitigate the evil effects of bad art; and the common result of a complete removal of fluid is, that the pleura in a few days fills up again, though in the meantime serious and irreparable damage may have been done to the lung.

But if we should be cautious about tapping

(1) Diseases of the Lungs and Pleura. Page 1026.

patients suffering from pleural effusion, who have previously shown a clean bill of health, we must be doubly careful how we interfere with an effusion complicating an established case of pulmonary tuberculosis, a pulmonary tuberculosis too, of which we may be quite ignorant: we may not have known the case before, and the previous symptoms may not have been very marked, or they may have been overlooked. Many injuries may be done to a tuberculous lung by aspirating a pleural effusion; but there are two great risks of an immediate nature which should never be forgotten, haemorrhage and pneumothorax. A case which I was able, while an undergraduate, to follow from the wards to the post mortem rooms impressed me very much.

J. F. admitted to Ward 29, Royal Infirmary, 16th December 1896. About a year before he had had a pain in the side. Since then he had slight cough but almost no sputum. Sixteen days before admission, that is to say on December 1st, 1896, he had a shiver with pain in the left side. Next day he saw a doctor. On the 4th December, the case was diagnosed as pleurisy. On the 14th fifty ounces of sero-fibrinous fluid were removed by tapping. He became collapsed at the end of the operation and on the 16th December, he was sent to Hospital. On admission he was found to be in a moribund condition and died in a few hours.

Post mortem report:- The left pleura contained much air and two pints of yellow purulent fluid. At the apex of the lower lobe of the left lung was a small irregular cavity with a passage leading into the pleural sac; the mouth of this passage was covered by recent lymph. The lung was quite collapsed. I think there can be little doubt that this patient died as the result of the operation of paracentesis. Such cases may be common enough; they would be certified quite truly as "Phthisis and Pneumothorax", and the real cause of death - paracentesis - would not occur on the certificate. Hence what Wilson Fox wrote in 1885 may still hold good: "If the proportionate frequency of sudden death in all cases of unrelieved effusions be compared with the number of cases in which it has occurred in all cases of paracentesis, the latter will be found to be vastly in excess of the former."⁽¹⁾

To summarize I have tried to show that if we have any means of saying definitely that a certain pleurisy with effusion is tuberculous, we ought not to perform paracentesis. If we have any means of saying that it is not tuberculous we need have no scruples. If it cannot be shown that we are able to diagnose the tuberculous from the non-tuberculous, then we ought, I think, to treat the case as an early manifestation

(1) Diseases of the Lungs and Pleura. Page 1052.

of tuberculosis, to treat it as we would an haemoptysis, to insist on a long convalescence of many months in the open air of the country with a more than abundant diet, and above all to abstain from tapping.

We must now once more return to the question: have we or have we not any means of diagnosing a tuberculous from a non-tuberculous pleurisy?

Many lines of enquiry suggest themselves as ones which might possibly be of value in aiding us to solve this problem; and I propose now to study some of these. They may be roughly divided into clinical and pathological. Let us take up the clinical first. The points to be studied in each case are:

1. The Family history.
2. The age of the patient.
3. The Temperature chart.
4. The duration and course of the illness.
5. The Physical Signs of Pulmonary Catarrh.
6. The State of the Glands.
7. The naked-eye characters of the pleural exudate.

1. The Family history. All questions involving an account of family history are difficult for many reasons, but chiefly for the cardinal one that very few people know accurately of what conditions their

relatives have died. Therefore from the very outset this source of information is tainted with suspicion. However, there is enough to show that this question of family history is not of much value in any special case, even where it can be settled. From private sources and from the insurance cases mentioned above, I have been able to obtain the family histories of 39 pleuritics. Of these, 23 ultimately became tuberculous and 16 did not. Of the 23 tuberculous, 14 denied any family history of tubercle, and 9 admitted to a near family history. Of the 16 non-tuberculous 4 admitted a family history and 12 denied it.

Hedges⁽¹⁾ again got a family history in only 23 per cent of the cases which ultimately developed tuberculosis: this is a much smaller proportion than my own cases show. As Harris and Beale⁽²⁾ have pointed out the personal element pervades this source of information so much that they actually found that 60 per cent of female patients in a consumptive ward admit to a family history of phthisis as against only 30 per cent of males: the reason being that in any family it is the women who know and discuss of what conditions their relatives die.

(1) St. Bartholomew's Hospital Reports for 1900.
Page 85.

(2) The treatment of Pulmonary Consumption. Harris and Beale. Page 52.

2. The Age of the Patient. To facilitate the study of this matter I may first refer to 21 cases of proved pulmonary tuberculosis which I have had the opportunity to study personally; all were in good health previous to the onset of an acute pleurisy. In all, I have myself found the tubercle bacillus, and all were quite clear and definite as to their history.

Secondly, I am permitted to give statistics of 47 cases which have been under the care of Dr William-son both in his private practice and in the Royal National Hospital; I am much indebted to him for the privilege of studying and quoting these.

The following is a list of my own cases.

No.	Name	Age at Pleurisy.	History of Pleurisy.	Sputum first seen.	Physical Signs.
1.	P.M.	33	Left pleurisy dry, March 1897. Left pleurisy with effusion. Sept. 1897. Not tapped.	October 1897. Tubercle bacilli found.	Both sides attacked
2.	J.R.	24	Left pleurisy with effusion 1892. Tapped. Right dry pleurisy one year before.	1897. Tubercle bacilli found.	Right lung attacked

No.	Name	Age at Pleurisy.	History of Pleurisy.	Sputum first seen	Physical Signs.
3.	J.D.	39.	Right pleurisy 1892.	Haemopty- sis in 1896. Tubercle bacilli found 1897	Both lungs attacked
4.	W.R.	4.	Left pleur- isy after wetting.	Ever since the pleur- isy. Tubercle bacilli present.	Left lung only attacked when 7 years of age.
5.	M.R.	21.	Right pleurisy 1892.	Sputum in 1897. Contains tubercle bacilli.	Left lung attacked
6.	J.S.	27.	Left pleur- isy 1895.	Cough ever since the pleurisy with sputum. In 1897 this contained tubercle bacilli.	Both lungs attacked
7.	M.M.	20	Right pleurisy with effusion in 1895 not tapped.	1897. Tubercle bacilli found.	Both lungs attacked
8.	W.Y.	37	Right pleurisy 1897. 7 weeks before cough & sputum.	1897 Tubercle bacilli found, <i>7 weeks after</i> <i>the onset of</i> <i>pleurisy</i>	Both lungs attacked

No.	Name	Age at Pleurisy.	History of Pleurisy.	Sputum first seen	Physical Sighs.
9.	G.S.	33	Right Pleurisy with effusion. Tapped. 9 months later sputum occurred.	1897. Sputum containing tubercle bacilli.	Right lung attacked
10.	M.C.	30	Right Pleurisy with effusion 1898. Not tapped.	1899. Tubercle bacilli present.	Right lung attacked
11.	A.C.	31	Right pleurisy with effusion 1896.	1898. Tubercle bacilli found.	Right lung attacked
12.	S.	24	Right pleurisy with effusion Not tapped	4 months after pleurisy. Tubercle bacilli found.	Right lung attacked
13.	F.	17	Left dry pleurisy 1899.	1900. Tubercle bacilli found.	Left lung attacked
14.	P.	30	Left dry pleurisy 1898.	1898. Tubercle bacilli found.	Right lung attacked

No.	Name	Age at Pleurisy.	History of Pleurisy.	Sputum first seen	Physical Signs.
15.	H.	31	Right pleurisy with effusion in 1884. Not tapped.	1900. Tubercle bacilli found.	Right lung attacked
16.	G.	43	Right pleurisy with effusion 1900. Tapped 80 oz.	Ever since the tap- ping Tubercle bacilli found.	Right lung attacked
17.	A.	26	Left dry pleurisy 1892.	1897. Tubercle bacilli present.	Left lung attacked
18.	M.T.	30	Right dry pleurisy 1900.	5 months later. Tubercle bacilli found.	Left lung attacked
19.	M.L.	16	Left pleur- isy with effusion tapped 15 months before I saw the case. Left empyema 9 months later. 1899.	1900 Tubercle bacilli present.	Both lungs involved

No.	Name	Age at Pleurisy.	History of Pleurisy.	Sputum first seen	Physical Signs.
20.	G.C.	31	Left pleur- isy with effusion. 1899. Not tapped.	1900 Tubercle bacilli present.	Right lung attacked
21.	M.S.	34	Left Pleur- isy with effusion. 1895.	1900 Tubercle bacilli present.	Left lung attacked

Of these cases the oldest was 43 at the time of the original pleurisy. Ten were under 30 years of age. Eighteen were under 35. The average age of all was 27. Pleurisy itself is not a disease which attacks young adults in so great a preponderance as this: so that a case occurring between 25 and 35 is more likely to be tuberculous than if it occur at any other time.

In Dr Williamson's 47 cases, four were over forty years of age and actually 36 were under thirty years of age. The average age in his list was about 26. These figures tend to confirm my own.

The practical result, however, of this enquiry is, that it does not give much help in any individual case. We can never be sure that a young pleuritic is tuberculous; never certain that an older one is not; so that we can never give the latter the

assurance that his advancing years give him freedom from the fear of tuberculosis, or from the irksome treatment, which that imposes on him.

3. The Temperature Chart. It would have been a very natural thought that here at any rate, we were pretty certain to get some indication of the true nature of the pleuritic process. A priori one would have said that unusual prolongation of pyrexia, especially of the remittent type would, after exclusion of empyema, have pointed in all probability to a tuberculous infection, actively proceeding. One would also have been tempted to regard a case with a short elevation of temperature as being either non-tuberculous, or at the least, as very quiescent if tuberculous.

Such views would receive support from the following type of case.

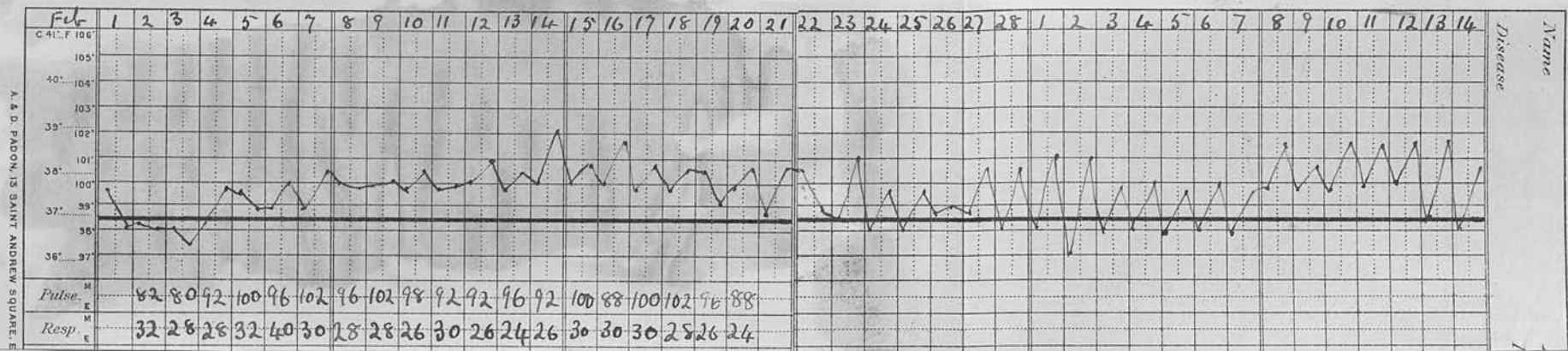
CASE I.

J. R. admitted to Ward 29 Royal Infirmary 1st February, 1898.

Complaint. Pain in left side, of a few days duration.

History. Left pleurisy with effusion five years ago - tapped. Dry right pleurisy one year ago. A few days ago was seized with present pain after chill.

Present State. Temperature as shown on chart.
Dry friction rub at left base.
Apices: nothing abnormal found.



The friction at the base continued for a long time to be the only physical sign in the chest, but it was felt that this was quite inadequate to explain the temperature. On February 22nd a few crepitations were heard at the left apex. On March 5th some sputum was obtained, tubercle bacilli were found, and the prolonged pyrexia was explained in the ways suspected.

In marked contrast to this, however, come two cases with prolonged pyrexia where there is reason to believe that this did not represent an active tuberculosis.

CASE II.

G. R. Admitted to Ward 29, 15th October, 1897.

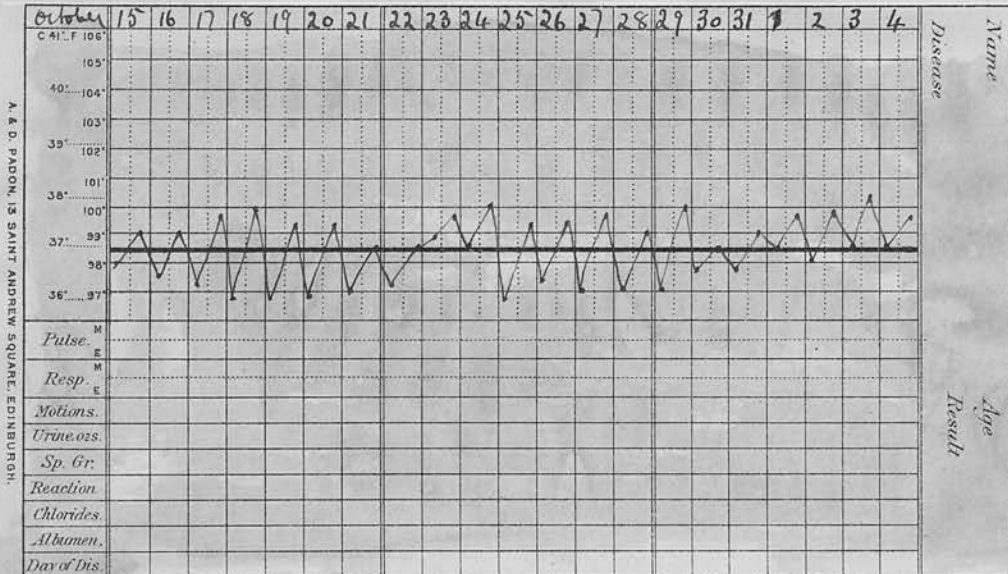
Complaint. Pain in the left side of the chest.

History. Six months ago had left pleurisy with effusion tapped "of a large amount". Kept well till a few days ago when he was seized with pain in left side of chest.

Present State. Temperature - intermittent febrile as shown on chart.

Signs in chest. There was some dulness and loss of expansion at the left base, due doubtless to the previous illness.

There was at present friction at both bases.



In about a fortnight the friction had disappeared, but the temperature did not settle and was certainly very suggestive of tuberculosis. In another week the patient went home feeling fairly well, but still febrile. Three months later he returned saying he had now cough with expectoration. No tubercle bacilli could be found, though it was felt that they must almost certainly be present. . Nothing more was heard of the patient till I looked him up in September 1900, three years and a half after his original pleurisy. I found him in good health. The cough

and expectoration had lasted a few weeks and then had disappeared. Except for the fact that the semi-circumference at the left base was half an inch less than at the right, I could detect no abnormal sign in the chest. This case not only illustrates the point that prolongation of pyrexia does not necessarily indicate tuberculosis, but also brings forward prominently how necessary the recognition of the presence of the bacillus is, before the diagnosis of the disease should be made.

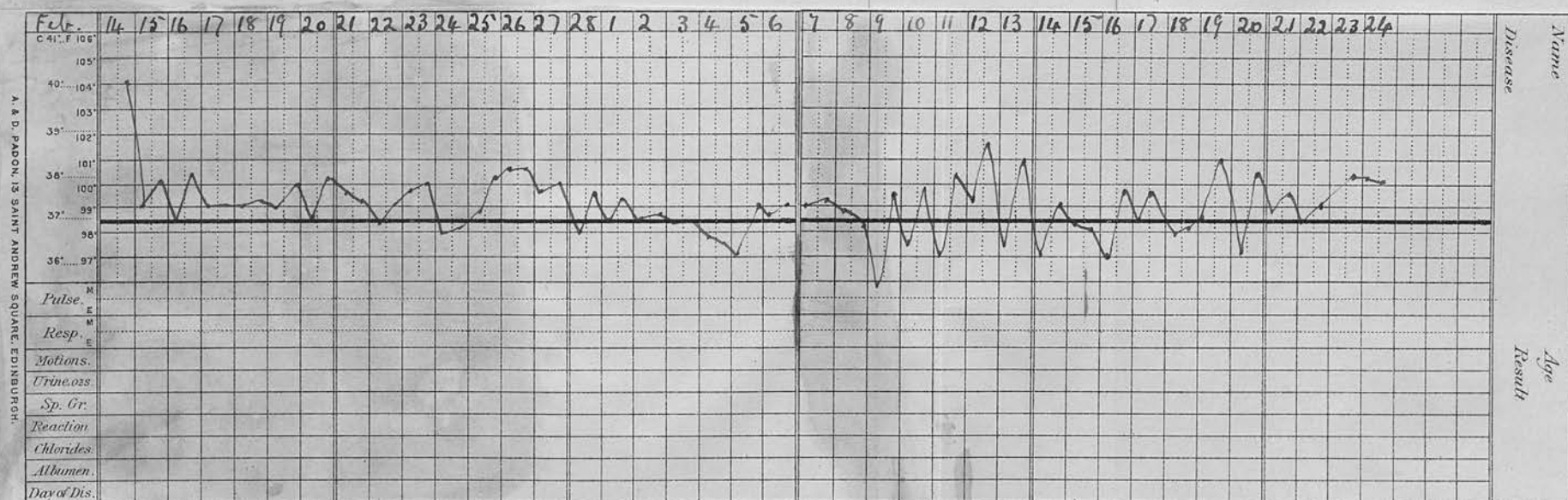
CASE III.

J. A. Similar to the last. Admitted to Ward 28 on 14th February, 1898.

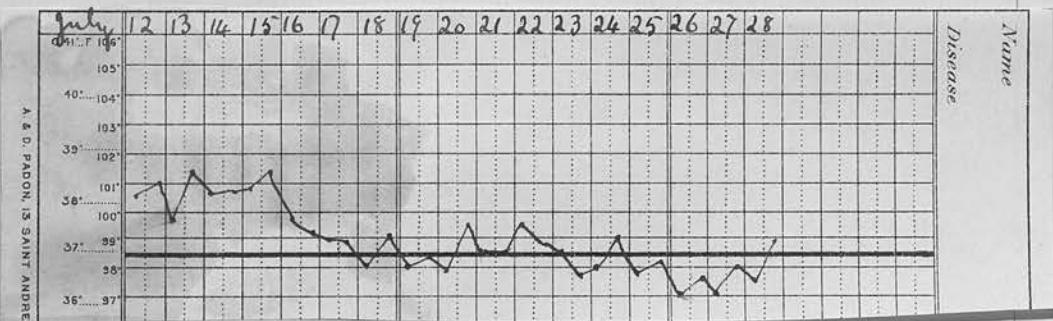
Complaint. Pain in the left side of the chest.

History. Quite well till November 1897, when got a chill, followed by pain in left chest. Put to bed and blistered. Kept well till three weeks ago when had a rigor and pain in left side. No cough.

Present State. Temperature as on chart. Signs in chest. An effusion up to spine of scapula on left side.



On February 23rd 1898, the patient was tapped of 30 oz. of serous fluid. On March 24th she went home in a state of pyrexia. In July she returned with a cough and some moist sounds in one apex and the temperature still irregular.



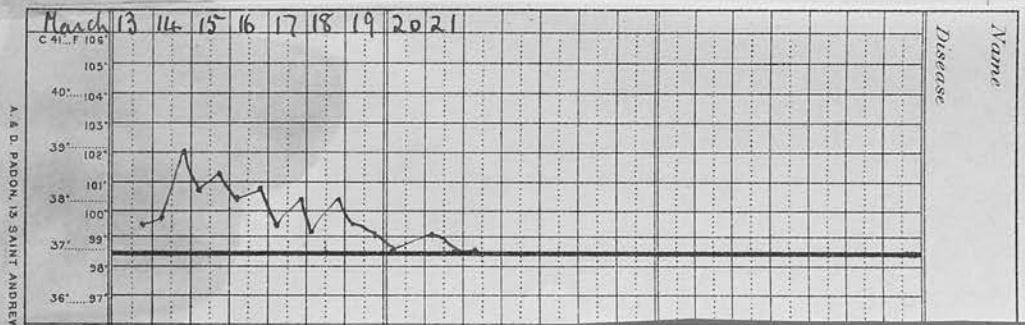
These signs, however, had disappeared before the 28th of July. No bacilli had been found. In August 1900 I found the patient to be quite well.

An enthusiast might say of both these last cases that they were cured cases of tubercle. Doubtless if one were advocating a cure it would be hard to keep them out of statistics. But in the course of a calm unbiassed study one sees that there is no proof whatever, that they were cases of tubercle; certainly they were not cases of active tubercle: so that one may justifiably argue that a prolonged pyrexia, even accompanied by cough and expectoration after an attack of pleurisy, does not point of necessity to the onset of tuberculosis, of active tuberculosis at any rate. We must now study one or

two charts where the febrile period was a short one, to see whether or no there by any relation between such a type and the absence of tubercle.

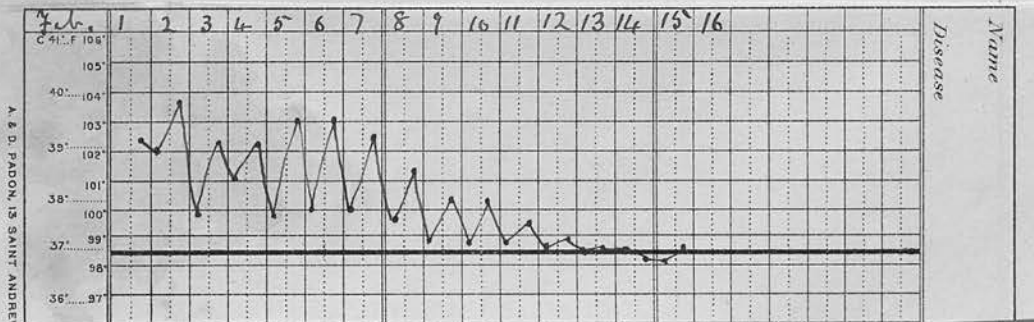
CASE IV.

F. K. on March 13th, 1891 was tapped of 23 oz. of clear fluid after an illness which had lasted three days. This patient is now (ten years later) quite well.



CASE V.

G. M. on February 1st, 1894 had pain in left side. This was due to friction. Effusion followed up to angle of scapula. It was not tapped. This patient is now (seven years later) quite well.



These two cases for which I am indebted to Dr Williamson's case book are in accord with a theory

that a short pyrexia is probably found with a simple case. Unfortunately no fixed law can be stated here either; as I have notes of a case of active tuberculosis, which quite upsets any such theory.

CASE VI.

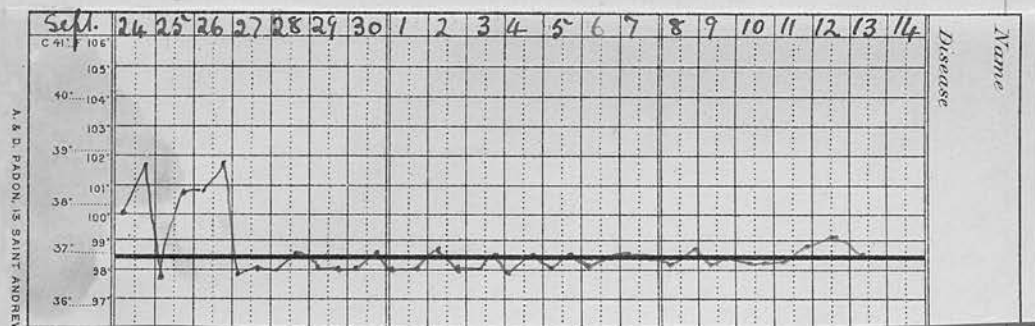
P. M. Admitted to Ward 29. September 24th, 1897.

Complaint. Pain in left side.

History. Dry pleurisy in March: ill only a few days, then well till a few days ago when had a shiver and pain in left side.

Present State. Temperature. Febrile for three days only. See below.

Chest. An effusion into left pleura which disappeared in a few days. Was left with cough and cracklings at apex. Tubercle bacilli found on October 12th.



The study then of these sets of charts shows us that we cannot predicate the presence or absence of tubercle from the type of the pyrexia.

4. The Duration and course of the illness.

Wilson Fox states that a long continued friction sound implies tubercle⁽¹⁾, also that tubercle tends

(1) Treatise of Diseases of Lungs and Pleura.
Page 1011.

to delay absorption⁽¹⁾. He adds, however, that these facts are not invariable, so that here again we have no absolute guide. In any case he states no time limit, so that we are left in the position that a prolonged case should arouse our suspicions.

Lately MacGuire has called attention to a form of disease where there is fever, emaciation and at first slight pleuritic rubs, multiple in number and flitting from place to place. This, he says, is soon followed by rapid consolidation and excavation of lung substance with a quickly fatal result⁽²⁾. This type of pleurisy should therefore be looked for.

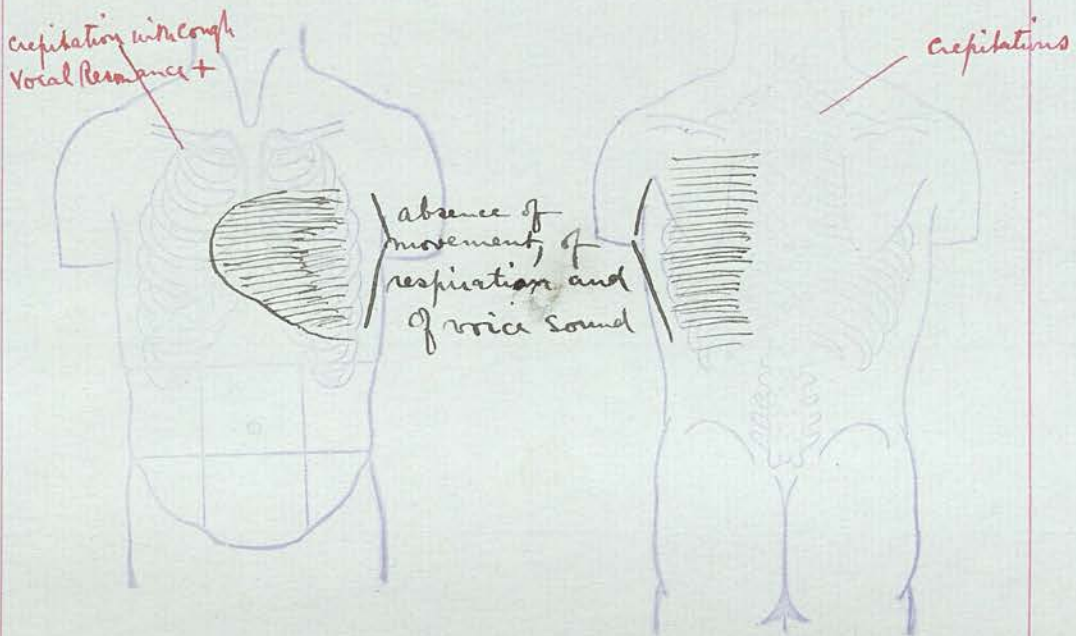
With this too, the question of relapses may be considered. It has certainly, in my experience, been a very common thing to get from pleuritics who ultimately become phthisical, a history of more than one attack of pleurisy. A series of relapses should make us doubly careful as to prognosis.

5. The onset of physical signs of pulmonary catarrh, at one apex after a pleurisy is not of necessity the indication of the commencement of a pulmonary tuberculosis. On this point the late Sir Thomas Grainger Stewart used to lay emphasis in his oral teaching.

(1) Ibid. Page 1025.

(2) The Harveian Lectures on Prognosis & Treatment in Pulmonary Tuberculosis by Robert MacGuire. British Medical Journal. Dec. 1st. 1900. Page 1566.

The case of J. A. (page 38) is one in point. This patient returned six months after a pleural effusion on the left side, with signs suggesting early infiltration of the right apex. These, however, entirely cleared off. The accompanying chart shows the state of this patient during the effusion (black), and subsequently six months after (red). She has now no signs of disease.



6. The State of the glands. Osler suggests that the presence or history of enlarged glands in the neck is of great importance, and says that: "infection⁽¹⁾ of the pleura in tuberculosis of these glands has been frequently noted."

(1) The Shattuck Lecture. Page 60.

7. The naked eye nature of the exudate. A haemorrhagic exudate is often viewed with suspicion, but it is certainly not uncommon even when there is no tuberculous element as in malignant disease and even in simple pleurisy.

It is seen then that by these various methods of investigation we are very far indeed from having any absolute or real guide on the important question as to whether or not any given case is tuberculous. They are not, however, utterly valueless. If a pleuritic confesses to a near family history of tubercle, is between 20 and 30 years of age, has high temperature of intermittent or remittent type and has been recently exposed to the infection of phthisis, we ought without doubt to say to such an one that he is threatened with pulmonary tuberculosis and treat him as such. It is folly to wait either for signs in the chest or bacilli in the sputum.

From this somewhat barren line of enquiry, which, however, it was well worth our time to pursue if only to obtain a negative result, let us turn to the pathological method, and ascertain whether any help is to be got there. Even, however, should this method prove to be of assistance, it cannot in actual practice be so useful as a successful clinical method

would be; because owing to the present paucity and poverty of laboratories its application must remain limited.

The methods employed are:-

1. To search the exudate for micro-organisms by staining or culture methods.
2. To inject the exudate into living animals.
3. The tuberculin test.

1. Searching the effusion for organisms. The attempt to find tubercle bacilli in the deposit of an effusion has been made frequently and almost always with a negative result. They are more readily found in cases of tuberculous empyema, as stated on page 18.

The attempt to grow cultures of tubercle bacilli also, has almost always ended in a negative result. If bacilli can be cultivated at all they will be found in the coagulated portions of the effusion⁽¹⁾. The most usual result, however, is that the effusion is found to be absolutely sterile⁽²⁾. While this would distinguish these cases from a pneumococcus or other organismal effusion, it does not help at all to distinguish tuberculous from "rheumatic" cases, and so is of little use.

2. Inoculation of animals. Netter⁽³⁾ practised

(1) Fowler. Diseases of the Lungs. Page 349.

(2) Osler. The Shattuck Lecture. Page 8.

(3) Netter. Bulletin des Hôpitaux. 1891. Page 176.

inoculation of guinea-pigs with the exudate of 20 cases of pleurisy apparently a frigore. In 8 cases tuberculosis developed in the animal. As a control experiment ~~he~~ injected into other 9 animals the exudate from 9 cases of pleurisy with effusion occurring as a complication of Bright's disease or rheumatism. In these no animal developed tuberculosis. As, however, he did not follow his twenty pleuritics up, to see what became of them, his observation remains incomplete and not of great value.

3. The Tuberculin Test. Netter⁽¹⁾ quotes from the reports of the Prussian Government as to this test. Fifteen pleuritics were inoculated and a positive result was obtained in thirteen. Unfortunately the ultimate event in the lives of these people is not recorded, so that again the experiment is of doubtful value, a value which is still further depreciated when we learn that at least 8.5 per cent of healthy individuals react positively to the test. Even, however, if the test were of value few patients would in the light of its history care to undergo it; an uncertainty as to diagnosis would be preferable to the possible lighting up of a general tuberculosis.

These latter pathological methods of enquiry have not then taken us much further than did the

(1) Quoted by Herbert B. Whitney. Twentieth Century Practice. Vol.VII. Page 11.

former clinical ones. It is however to be remembered that they are still in their infancy, and may be expected to produce better results later. We are at present left in the position that it must be very seldom that we can assert that any given pleurisy is not tuberculous; and therefore, though we believe that the majority of them are not, it is our duty to lay the possibility of such an event before the patient. To wait for certainty of diagnosis is to rob the patient of any chance of being cured.

PART II.CERTAIN OBSERVATIONS ON CASES OF PULMONARYTUBERCULOSIS WHICH STARTED FROM APLEURITIC ORIGIN.

Anyone who has studied the modes of onset of pulmonary tuberculosis must have remarked how difficult it is for the patient, in the majority of instances to state with precision when he was last quite well. The disease usually begins insidiously. There is a cough off and on for months, or the patient has had a cough for several winters, but it stayed on into last summer, or he has been losing weight of late, or been sick in the morning or simply has been feeling below par. But although this is the common story; a certain number of patients can give a definite date up to which they were well and strong, and since which they have been ill. Sometimes this history is one of sudden haemoptysis; sometimes it is of an influenzal attack; sometimes we are told of a sudden pain in the side with hard dry cough lasting for a few days, and which may or may not have been diagnosed as pleurisy: at any rate the full measure

of health was never regained; there has remained cough ever since. Still a third class of patients report the onset of some definite illness followed by apparent recovery for a period of months or years; and then cough sets in either insidiously or after some additional cause of lowered health, such as an influenzal attack. To each of those classes our pleuritics belong; but it is only in the second and third that we are able to deal with them in a satisfactory way.

To follow this into detail, I have been able to analyze the histories of the onset of pulmonary tuberculosis in 532 persons. I have myself seen 77 of these, 35 while resident physician to Dr Affleck, in Edinburgh, the remainder in Ventnor as assistant to Dr Williamson. I am indebted to these gentlemen for the great privilege of using the information which I was then able to gather. Of those remaining, 329 are from the private case book of Dr Williamson, and 126 from his hospital case book. I am indebted to him for permission to quote these cases.

Out of these 532 cases, a clear history of pleurisy was obtained in 76 or 14 percent.. This is a larger proportion than that given by James who says that ⁽¹⁾"out of 170 cases in which the pers-

{1) Pulmonary Phthisis. James, 1888. Page 59.

onal history had been carefully enquired into, certainly 9 had suffered from and ascribed their illness to an attack of pleurisy."

In this same list of 532 cases there were 86 who attributed the disease to an attack of haemoptysis, i.e. that was the very first symptom. It is curious to contrast this with the 76 pleuritics, when we consider how recently pleurisy has been regarded as a forerunner of phthisis, and how long haemoptysis. So recently indeed has this grave view of pleurisy become specially prominent that in 1886 Douglas Powell wrote⁽¹⁾: "Among the common causes of simple pleurisy, exposure to cold is the most frequent Certain diathetic states favour the occurrence of such pleurisies, e.g. the rheumatic or albuminuric." This, however, is one of the last publications putting forward the "a frigore" view: in 1888 Fagge said:⁽²⁾ "It may be said that pleuritic effusion is in old persons more often due to renal disease, and in young persons to tubercle."

In order to emphasise still more the frequency of the previous history of pleurisy in cases of pulmonary tuberculosis, I give a table in which the various chief modes of onset are contrasted.

(1) "On diseases of the lungs and pleurae". by R. Douglas Powell, 3rd Edit. 1886.

(2) "The Principles and Practice of Physic" by Hilton Fagge, 2nd Edit. Vol.II. page 178.

	Arising from Pleurisy	Arising from Haemorrhage	After Influenza	After Pneumonia	Commencing Insidiously.
Dr Williamson's Private Cases.	31	58	35	8	197
Dr Williamson's Hospital Cases.	24	23	17	6	56
Cases seen by self in Edinburgh	9	1	7	2	16
Cases seen by self in Ventnor.	12	4	4	1	21
Total 532.	76	86	63	17	290.

As this table shows, haemoptysis and pleurisy are almost equally common as a starting point in the symptoms of tuberculosis. Indeed, it is a question whether it be a more ominous thing for a man to have an haemoptysis or an attack of pleurisy. I have no figures to show; but I believe that a larger number of those who have had pleurisy ultimately suffer from tuberculosis, than of those who have had haemoptysis.

It is a point of much interest to consider on which side after a pleurisy do we find the subsequent pulmonary condition. Naturally, we would expect it on the same side, but such is not always the case. Dr James gives three cases in which the phthisis,



seemed to begin on the same side, ^{as the pleurisy} and three in which it seemed to begin on the opposite. ⁽¹⁾ As regards my own cases, as reported on pages 30 to 34, there are seven cases of pleurisy with effusion where the tuberculosis appeared on the same side as the pleurisy. These cases are Nos. 9, 10, 11, 12, 15, 16, 21. One case of pleurisy with effusion was followed by a tuberculosis of the opposite side, No. 20. Among the dry pleurisies ~~these~~ ^{three} were followed by tuberculosis on the same side, Nos. 4, 13, 17. Three were followed by subsequent disease on the opposite side, Nos. 5, 14, 18.

The fact that the pulmonary sequel does sometimes appear first on the lung of the other side has a bearing on what has been already said as regards paracentesis thoracis. It is maintained by some that we should tap early, lest the impaired activity of the lung resulting from its prolonged compression should be the cause of its becoming the seat of a tuberculous process. If this were the danger it is obvious that the pulmonary tuberculosis ought always to occur on the same side as the pleurisy; but it does not. There is of course the further fact that even when the disease does occur on the same side,

(1) Pulmonary Phthisis. Page 60.

it is apical, whereas in pleurisy with effusion the greatest amount of compression and consequently the greatest amount of functional inactivity are basal. As stated on page 22, it is not likely that pleurisy is to be regarded as a predisposing cause of tuberculosis, but that in cases where it is followed by the latter, it is really a manifestation of its actual presence in the lungs or pleural membranes themselves.

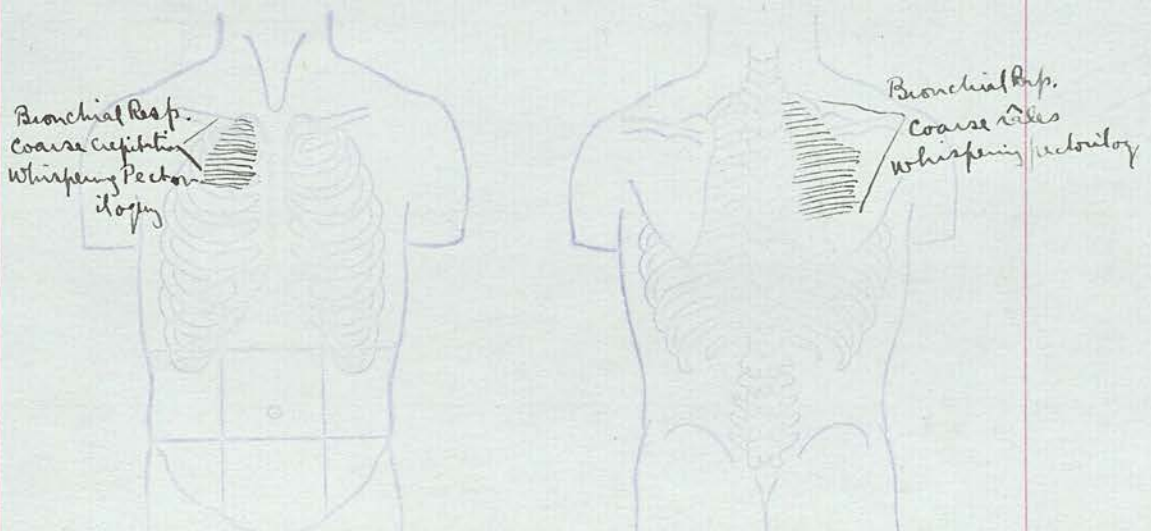
It has been frequently urged in the preceding pages that the pleural effusion is best looked on as a great and special reaction by a healthy organism against an attack of tubercle. If this be the case, we should expect that cases of pulmonary tuberculosis following after a history of pleurisy would be very chronic in their nature, and would above all others tend towards recovery. I hope to show that this is so.

First, there is commonly an interval of time elapsing between the pleurisy, and the onset of clinically recognisable signs of pulmonary infiltration. In other cases of phthisis we do not find this as a rule. On the contrary the patient progresses -- speaking pathologically -- from bad to worse. Here, however, after a primary manifestation of a tuberculous process, we have a pause, during which as it were the organism gets a chance of righting itself.

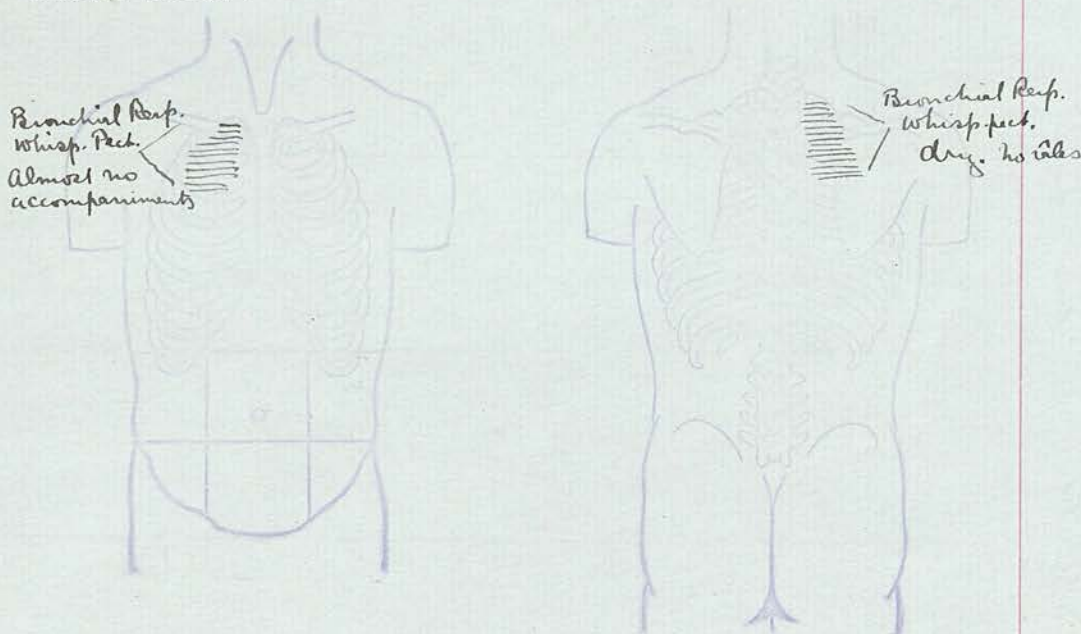
Secondly, one can quote a number of cases of phthisis, all of which were very chronic and benign in their course, in the case of those who were old pleuritics. I cannot show many cases of my own, as the majority of those which I have seen have been watched for short periods of time only, either while passing through a hospital ward, or on a short visit of a few months to Ventnor, during my two and a half years of work there. Of three cases I can speak personally. I have no hesitation in saying that they are the most successful cases of pulmonary tuberculosis with which I have come in contact. A fourth case, which died after a slow course, I will refer to later, as it was of importance from the point of view of morbid anatomy.

Case 1. M. P.:

Had pleurisy in 1898. In April 1900 had right apical tuberculosis - tubercle bacilli present. His state then was as on the following diagram. His weight was 9 stone, 4 lbs.



In February 1901 his weight was 10 st. 10 lbs. and his chest was much freer from accompaniments as shown below.



Case 2. M. C.:

Pleurisy in October 1897. In November 1899 there was cough and expectoration which contained tubercle bacilli. At both apices there were a few crepitations. In July 1900 while the crepitations could still be heard, when the patient coughed, there had been an entire absence of expectoration for one month.

Case 3. W. R.

Left pleurisy in 1890, followed by left phthisis. In 1897 had signs of a large dry cavity in left lung; but the general health was quite well.

Out of the cases occurring in Dr Williamson's practice from the years 1885 to 1897, the subsequent history has been obtained in nine. These are sub-joined in tabular form.

Name	Pleurisy	{side & date.	Onset of Phthisis.	Result.
1. Mrs M.	Right	1884	Right 1884	Died 1899.
2. Miss B.	Left	1884	Left 1885	Apparently recovered 1895
3. Miss S.	Right	1886	Right 1887	In statu quo 1890.
4. Miss P.	Left	1888	Right 1889	No symptoms 1894.
5. Mr S.	Left	1881	Left 1885v	Died 1894.
6. Mr F.H.	Left	1888	Left 1888	Apparently recovered 1893
7. Mr C.	Left	1894	Left 1894	Died 1895.
8. Mr K.	Left	1891	Both 1893	Died 1895.
9. Mr N.	Right	1889	Right 1891	No symptoms, 1900.

This table is sufficiently remarkable in that it is able to show four cases of virtual cure. Of these, No.2 and No.6, had at the dates mentioned neither symptoms nor physical signs of disease. The other two, No.4 and No.9, were without cough or sputum. As regards physical signs they still had dulness with harsh bronchial breathing at one apex - signs however

which do not entail the actual presence of an active tuberculous process. Of the four cases which were terminated by death, two deaths occurred at intervals of more than twelve years after the initial symptoms; and in the whole set of cases two only displayed any rapidity in their downward course.

Of course, these cases are not sufficiently numerous to prove anything; but they are suggestive; and if in a larger number these facts hold good, we may be able to enunciate the important proposition, that in tuberculous cases a previous pleurisy, far from having predisposed to the tuberculosis, is to be regarded as an initial sign of favourable omen in the history of the present disease.

Practically then, it might be well to seek out such history of previous pleurisy in our cases of pulmonary tuberculosis; because if we find it we might be able to congratulate the patient on his chances, and assure him that in the past he had made a good fight against the morbid condition.

Whether a pleurisy with effusion, occurring in the course of a case of well established pulmonary tuberculosis, is to be regarded as a fortunate event or not, is a question of interest. Many writers say that it is favourable. Louis says that it accelerated the course of the disease. The

complication of sero-fibrinous pleurisy in the course of a phthisis is rare; and I have not personally had the advantage of seeing any case save the one described on page 26, where a fatal event ensued after paracentesis. With the idea that these effusions might be beneficial, it has been frequently proposed to inject saline solution into the pleural sac. On one occasion, at least, this has been done with a fatal result. When examined the procedure does not seem to be based on really scientific lines. Firstly, adhesions might be torn: even if the fluid were injected at a very low pressure, by its accumulation in the cavity this pressure would soon be much increased, and be very little under the control of the operator. Secondly, fluid thrown into serous cavities does not behave like fluid thrown out by living cells; instead of remaining like the latter for a period of days, weeks or months, it is rapidly absorbed and the status quo is soon restored.

Dry pleurisy can certainly be not other than salutary. As has been very frequently pointed out, ^{but} it is the great factor which prevents almost any case from running the risk of pneumothorax. It performs, however, another highly important service, which is well shown in the accompanying plate. This was drawn for me from a preparation which I made from a



tuberculous lung. The case was one originating in pleurisy and running a slow course. Post mortem, the pleura was found to be adherent almost universally. A portion with underlying lung was hardened in corrosive sublimate, embedded in paraffin, cut with a microtome, and stained with picro-carmin. It was mounted in Farrant's solution, and placed in a magic lantern slide. The image of the section was thus thrown on to the paper and was traced over with pigments while thus arranged. A permanent record was in this way obtained, which magnified the section to about fifteen diameters. The section shows the deep layer of pleura to be represented by a dense fibrous band from which run inwards other dense bands, which are obviously thickened interlobular septa. These, in their turn, are connected with other dense bands which form the lining wall of a cavity. These last are formed partly no doubt by new formation, but are also in large amount thickened interlobular septa, whose lobules have already been destroyed. The special point I wish to bring out is, that by a pleuritic process the pleura has become a strong fixed basis, from which the thickened interlobular bands may run to contract on the tubercular foci, and envelope them. If the pleura were not adherent these interlobular bands would not be

able to expend their whole contractive power on crushing out the tuberculous process, but would expend part of it on that, part of it on pulling the pleura inwards towards the cavity; and thus much of their curative power would be wasted. How strong this pull may be is well shown in the scoliotic backs, indrawn sides and displaced viscera of old cases of pulmonary tuberculosis which have done well.

Let me now in conclusion pass in review the main arguments which have been put forward in these pages.

We saw that notwithstanding their assertions to the contrary, modern writers have failed to prove the disappearance of pleurisy a frigore from the list of diseases. Indeed, it was shown that probably in only 25 to 35 per cent. of cases of pleurisy with effusion did pulmonary tuberculosis subsequently develop; that if it were going to develop at all, it would do so within ten years; and that thereafter the probability of an old pleuritic becoming the subject of pulmonary tuberculosis was in truth very small indeed. The importance of this as regards life insurance was insisted on.

In view of the idea that the treatment of a tuberculous pleurisy ought theoretically to be very different from a non-tuberculous one, the attempt was made to find some sign or symptom, which should be pathognomic of the ^{one} condition or the other.

Many lines of enquiry were suggested and followed up so far as our material took us; but the result was disappointing: our study led us only to this, that there is no symptom or sign, or set of symptoms or signs, which can give us in any given case, the confidence to pronounce such case either tuberculous or non-tuberculous. Therefore, as the tuberculous cases are of such grave import, as tubercle is a disease curable in its very earliest stages, as the treatment for it is not other than beneficial in the non-tuberculous cases, it seems to me that our plain duty, when called to such a case, is to point out to the patient or his advisers, the impossibility of ^{stating} the ultimate prognosis, and to insist that the patient should be treated as one suffering from incipient phthisis. We should do this even though we know that the odds are that any one given case will not become tuberculous.

Another duty of a negative kind we owe to such a case. It is that except when life is threatened, we are to refrain from the operation of paracentesis thoracis. There is a fascination about the opera-

tion which makes it hard for us to hold our hand. The patient has been told that he has water in the side; day after day it is still there; why is nothing being done to take it away? Then one day the physician taps, and the patient feels pleased, feels that now he will get on, that he never could have got on till all that water was gone; and the doctor acquires an additional credit in that household. Both parties are pleased, but we have seen what lamentable and irremediable damage may have been done to the patient.

The other reasons for paracentesis, viz. the fear of scoliosis, a permanently compressed lung or that tubercle might develop in an ill-functioning portion of pulmonary tissue, were found either not to be founded on fact, or to be of minor importance.

From this we proceeded to discuss the modes of onset of pulmonary tuberculosis, and saw that a considerable number of cases -- 14 per cent -- could trace their first symptom to a pleurisy. This was shown to be a question not only of academic interest, but of the greatest possible consequence to the patient, because we believed that these cases tended to become more chronic than any other, that their pleuritic history pointed to an ability, which they possessed above other patients, to fight the disease.

Lastly, as to the effects of pleurisy in the

course of phthisis. Pleurisy with effusion we saw was rare and its effect disputed. Dry pleurisy was universal and its effects not other than beneficial, firstly, in the prevention of pneumothorax, secondly, in the provision of a strong basis of support for the cicatricial bands which are to encapsule the diseased foci, and prevent further dissemination of the morbid condition.

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